

To make a practical suggestion, I would suggest that the physicians work for a measure in our laws which will require the court to submit medical questions to a board of physicians whose determination shall be final on those questions, and that they shall be employed and selected by the court and never appear as partisans. Until that time comes perhaps you can, by popular agitation, create a sentiment that the court shall select the medical experts and that you will not be employed on either side in such a controversy.

There are many other features of this subject which I would be glad to discuss with you and hope at some future time I may have an opportunity. These ideas are jotted down roughly and hurriedly with the idea of suggesting but the one thought, *elimination of partisanship*.

Very respectfully yours,

J. D. FREDERICKS,
District Attorney Los Angeles County.

Los Angeles, Cal., March 18, 1910.

Dr. A. S. Lobingier,

Chairman of Committee on Medico-Legal Expert
Testimony, of the County Medical Association,
Los Angeles.

My Dear Sir: Before I received notice that the proposed discussion of medico-legal expert testimony at a meeting of your society would occur this evening, I had made an engagement which calls me out of the city. This deprives me of the pleasure of being present at your meeting.

It is a principle common to all reforms that they must be preceded by a realization that the existing conditions are in some way wrong or defective. With respect to medical expert testimony, it is now well understood, in both the medical and the legal professions, that the present methods of selecting expert witnesses, and the present methods of obtaining their opinions as such experts, are very unsatisfactory.

They are unsatisfactory because they are not well adapted for promoting the objects for which legal investigations are instituted. Those principal objects, it scarcely seems necessary to say, are the ascertainment of truth, and the application of truth toward the ends of justice.

It is manifest to all who have given the subject close attention, that professional men, in giving their opinions on the subjects upon which they have exceptional knowledge and experience, should be as completely without bias or prejudice when they give their opinions in a court of justice, as they would be in applying or stating those opinions in consultation or practice in their own offices or at the bedside of a patient. In matters of opinion such as those here referred to, a court does not want the arguments of a partisan. What the court really needs is the special finding of a professional referee or judge chosen to give his opinion upon the matter, because it is supposed that in no other way can the truth be so well ascertained in that particular instance. Therefore, if a so-called expert is known to have been summoned and paid exclusively by one

party to a case in court, that fact has a tendency to detract from the confidence with which his evidence is received; and in its nature, also has a tendency to create such bias in the mind of the witness that, either consciously or unconsciously, he becomes less able to testify impartially with respect to the matter upon which his opinion is to be given.

It is gratifying to all who are interested in the right administration of justice in our courts, that special attention is now being given to obtaining changes in our laws with respect to this subject. By united action on the part of the medical societies and the bar associations of the state, public opinion may be roused and legislative action may be obtained, all moving in the right direction.

I trust that some new legislation, similar to that which has been proposed in the State of New York (but which, so far as I know, has not yet been adopted), will be prepared during the current year, so that it may be submitted to the next session of the California legislature. I am sure that in actively interesting yourselves in this question you are devoting your attention to a very important interest, not only of your profession, but of the people of the state.

With great regret that I cannot be present at your meeting this evening, I am,

Sincerely yours,

N. P. CONREY.

BOVINE TUBERCULIN IN THE TREATMENT OF PULMONARY TUBERCULOSIS.*

By WILLIAM C. VOORSANGER, M. D., San Francisco.

This paper is presented in the nature of a preliminary report, since the number of cases reported as treated with bovine tuberculin might be considered too small upon which to base definite conclusions.

In the *Lancet* of February 15, 1908, Nathan Raw reported successful results in eighteen cases of pulmonary tuberculosis treated with bovine tuberculin and a hundred and four case of surgical tuberculosis treated with the human form. Before the tuberculosis congress, held in Washington in October, 1908, he again reported glowing results in twenty-seven cases (no doubt nine added to the first eighteen) of pulmonary tuberculosis treated with the bovine form of tuberculin. Twenty-three of his cases were in the early stages and four well advanced, two of the latter even becoming apparently well with disappearance of all physical signs of the disease. In conclusion, Raw states: "From this series of cases of phthisis pulmonalis, I am convinced beyond the shadow of a doubt, that the tuberculin prepared from bovine sources has a most marked and curative effect in tuberculous affections of the lungs. . . . At present, I am quite certain that, under suitable conditions, every early case of phthisis should be inoculated with bovine tuberculin."

I was so impressed with Raw's results and his

* Read at the Fortieth Annual Meeting of the State Society, Sacramento, April, 1910.

enthusiastic statements, that I immediately began treating my cases of pulmonary tuberculosis with bovine tuberculin. Before presenting these results, let us ascertain if there is a rational working theory to justify a statement that this form of tuberculin has greater curative power than any other. Unfortunately, we are again working in the realm of empiricism, as we have in the past done with all tuberculins. We are again face to face with the dogmatic statement of Koch, made in 1901, and emphasized in 1908, as to the dissimilarity of human and bovine tuberculosis. The best scientists of all countries have challenged this statement and an immense amount of scientific investigation has been the result of the argument. Out of these investigations, by such men as Raw, Woodhead, Aufrecht, Ravenel, Arloing and Courmont, Wasserman and a score of others, I firmly believe came the inspiration to use bovine tuberculin as a curative agent. In 1908, Pochin reported some interesting observations by which he tried to ascertain whether the action of opsonins in bovine blood was the same with regard to tubercle bacilli of both human and bovine strain or whether differences did exist, which would indicate a distinction between bacilli with regard to their susceptibility to the action of opsonins and, therefore, a difference in the resistance in the animal to infection by the bacilli of different strains. He found that marked differences did exist. Out of such experiments as these can be evolved not a theory, but an hypothesis, that if we accept as true that pulmonary tuberculosis is caused by the bacillus of the *typus humanus*, and gland, bone, joint and other forms of surgical tuberculosis by the *typus bovinus*, the opposite strain should always be used in treatment; that is, human tuberculin in the bovine form of tuberculosis,—bovine tuberculin in the human form. I grant you that we are yet far from a working theory, which will solve the question of tuberculin immunity. There is no unanimity of opinion—each one is experimenting—each experimenter believes his methods the most satisfactory.

In a recent publication, I collected the various tuberculins on the market, to the number of seventy. No doubt, this number has since been augmented, and each tuberculin has one or more successful champions, who report successful cases. Spengler, an able scientist, will claim in his *I. K. or Immun Korper* (body) almost the tuberculin millennium, and still German literature abounds in reports of failures by those who have used it. Therefore, until the question of tuberculin immunity is solved, and until we really know what the real action of tuberculin is, we can not altogether condemn any investigator's method as futile. We should be good skeptics but reluctant scoffers. We should not forget that in tuberculin we have a toxin potent for good or evil effects. When we work with an antitoxin, as in diphtheria, we are injecting a substance which contains antibodies, call them agglutinins, precipitins, or by what name or numbers of names you will; there are many of them which have crept into the nomenclature of serum therapy, just so often as some investigator enunciated a supposedly new theory. Now, when we work with a toxin,

such as tuberculin, our aim is to rouse to action the natural defenses, already in the body—which we will simply call antibodies, so as to avoid confusion. As I worked along with bovine tuberculin, and waded through the almost endless and ever continuous literature, upon the general subject of immunity, and tuberculin immunity in particular, it occurred to me that a tentative theory might be evolved based upon a small but rather successful series of results. It does seem possible that if there is more curative power in bovine than in other forms of tuberculin, it can only occur through the production of a greater number of antibodies. In a discussion of my results, I believe that I can demonstrate a slight justification for making this statement.

Up to date, I have treated twenty-eight cases of pulmonary tuberculosis with bovine tuberculin, using at first the B. F. and afterwards the T. R., believing the latter the most efficacious, since the entire bacillus is used in its production. These twenty-eight cases were picked from a material of over one hundred cases of tuberculosis occurring in clinical and private practice. I have drawn up a tabulated report of these cases which shows that twelve are incipient, thirteen moderately advanced and three far advanced. You will notice that the classification shows as many moderately advanced as incipient cases. I believe, that we are prone to call a case of tuberculosis incipient when in reality the lesion is quite well advanced. Flick is a little too conservative, when he states that the incipient stage of the disease is the stage before symptoms manifest themselves. If this were true incipient cases could only be discovered by accident. But any one who has worked much in the field of pulmonary tuberculosis will agree with me that no case where physical signs are fairly manifest, can in truth be called incipient. The X-Ray has proven this beyond a question of doubt. The three far advanced cases were treated by way of experimentation to ascertain if any kind of result could be obtained with them. One was slightly improved; two died. This shows again that any form of tuberculin, to be effective, must find a responsive medium. Where resistance is low, antibodies will not be formed. If the organism has lost its power to react, then the use of tuberculin will prove inefficacious. It should not be used for this reason in advanced cases.

Of incipient cases, nine were right apex involvement; three apex. Of the moderately advanced, in five cases both apices were involved; in five cases part of one or both upper lobes; in one case, right upper lobe and left apex; in one case, upper lobes of both lungs, and in one case middle lobe of right lung. The number of injections varies and demonstrates some interesting facts. They range from twenty-six to fifty-eight; the best results were obtained with the fewest injections. Those receiving a large number of injections where improvement did not begin fairly early showed but slight or no improvement. The period of treatment ranged from approximately two and one-half months to one year. The most brilliant result was obtained in a man, aged thirty-six years, with lesions of both apices who

Patient.	Diagnosis.	No. of Injections.	Period of Treatment.	Reaction.	Result.	
S. M. 38 yrs.	Incip. rt. apex bacilli pres.	30	Aug. 24-09 Feb. 1-10	None	Apparently cured. 10 pounds.	Gained
B. L. 25 yrs.	Incip. rt. apex Bacilli not present.	27	April 17-09 Aug. 15-09	None	Apparently cured. 8 pounds.	Gained
H. K. 24 yrs.	Mod. adv. middle lobe rt. lung bacilli pres.	38	Aug. 4-09 Jan. 25-10	None	Apparently cured. 18 pounds.	Gained
M. K. 27 yrs.	Far advanced bacilli pres.	58	April 9-09 Nov. 9-09	June 2-09 Aug. 15-09 Oct. 4-09	Died.	
M. H. 36 yrs.	Mod. adv. both apices bacilli pres.	40	Nov. 15-09 April 1-10	None	Apparently cured. 26 pounds.	Gained
L. L. G. 30 yrs.	Incip. rt. apex bacilli pres.	51	May 12-09 Jan. 16-10	May 25-09	Arrested. Gained 10 pounds.	
R. G. 45 yrs.	Mod. adv. rt. upper lobe—L. apex bacilli pres.	12	March 12-10 April 15-10	None	Improving. Gained 8 pounds. Still under treatment.	
R. E. D. 38 yrs.	Mod. adv. rt. upper lobe bacilli pres.	26	June 18-09 Sept. 15-09	None	Improved. Gained 9 pounds. Ceased treatment.	
F. C. 51 yrs.	Incip. rt. apex bacilli pres.	32	Aug. 25-09 Feb. 1-10	None	Arrested. Gained 10 pounds.	
W. G. B. 25 yrs.	Mod. adv. rt. upper lobe bacilli pres.	41	Jan. 21-09 Aug. 2-09	None	No improvement. Treatment suspended.	
J. A. 49 yrs.	Incip. rt. apex bacilli present.	44	May 2-09 Dec. 6-09	None	Apparently cured. 18 pounds.	Gained
B. L. 23 yrs.	Mod. adv. rt. upper lobe bacilli present.	29	Dec. 5-09 April 15-10	None	Arrested. Gained 12 pounds.	
A. F. 27 yrs.	Incip. rt. apex bacilli pres.	8	March 15-10 April 19-10	None	Improved. Gained 6 pounds. Still under treatment.	
J. F. 30 yrs.	Incip. rt. apex bacilli pres.	8	March 15-10 April 19-10	None	Improved. Gained 5 pounds. Still under treatment.	
V. N. 43 yrs.	Mod. adv. both apices bacilli pres.	28	Dec. 30-09 April 15-10	None	Arrested. Gained 18 pounds.	
J. M. 19 yrs.	Incip. rt. apex—bacilli pres.	29	June 28-09 Sept. 3-09	None	Apparently cured. 10 pounds.	Gained
W. P. 36 yrs.	Mod. adv. upper lobe rt. lung—bacilli pres.	18	Sept. 16-09 Nov. 22-09	Slight Nov. 1-09	Slightly improved. Sent to Alta.	

E. L.	46 yrs.	Mod. adv. upper lobe rt. lung L. apex—bacilli present.	31	May 6-09 Sept. 16-09	None	No improvement. Left town.
J. S.	23 yrs.	Mod. adv. both apices—bacilli present.	23	May 6-09 Sept. 16-09	None	Apparently cured. Gained 19 pounds.
A. K.	31 yrs.	Incip. rt. apex bacilli present.	27	May 6-09 Oct. 7-09	None	Apparently cured. Gained 20 pounds.
C. A. E.	48 yrs.	Incip. left apex bacilli not present.	51	May 6-09 Jan. 6-10	None	Apparently cured. Gained 5 pounds.
L. M.	20 yrs.	Incip. left apex bacilli present.	19	Nov. 18-09 Jan. 20-10	None	Improved. Gained 5 pounds. Left town.
B. G.	27 yrs.	Mod. adv. upper lobes both lungs bacilli pres.	53	July 8-09 Feb. 17-10	Nov. 27-09	No improvement. Lost 6 pounds.
K. P.	29 yrs.	Incipient left apex bacilli pres.	26	Jan. 13-10 Feb. 16-10	None	Improving. Gained 5 pounds. Still under treatment.
P. V. S.	40 yrs.	Mod. adv. both apices bacilli pres.	44	June 5-09 Feb. 1-10	None	Arrested. Gained 10 pounds.
M. R.	40 yrs.	Far adv. bacilli pres.	20	May 5-09 Aug. 1-09	None	Slightly improved.
H. S.	38 yrs.	Mod. adv. both apices bacilli not present.	28	April 15-09 Jan. 20-10	None	Arrested. Gained 14 pounds.
J. N.	41 yrs.	Far adv. bacilli pres.	12	March 20-09 June 25-09	April 2-09 May 15-09	Died.

received in all but forty injections, during a period of four and one-half months. After two months, all symptoms disappeared, including cough and sputum and he had gained twenty pounds in weight. As a precaution, I continued giving the injections for two and one-half months longer, or until April 1, 1910, when I discharged him as apparently cured and was gratified to hear him state that he never felt better in his life. His total gain of weight was twenty-six pounds. Needless to say, his case was verified by the finding of tubercle bacilli in the sputum.

During the treatment of this series of cases, I had reactions in five cases. In two very slight, in three, quite pronounced. Observers are agreed that we should avoid reactions and if our cases are chosen properly, and if one starts with a low enough dose, and increases very gradually, there is no question but what reactions can be avoided. It did seem to me that where I obtained reactions they were rather more violent than those I had seen in use of human tuberculin.

The preparation and dosage of bovine tuberculin is important as it is with all forms of tuberculin. I am now using the T. R., and have it prepared according to prescription at the Cutter Laboratory. I start with 0.1 cc. of 0.0001 solution or a 1/50000

mg. of solid substance. This is always my minimum dose. I have no maximum dose, since I do not believe that it is yet proven that there is an actual condition of tuberculin immunity and I aim only to produce a tuberculin tolerance. The latter varies naturally in different patients, and the concluding dose is based upon the patient's condition and his tolerance. When symptoms have disappeared, I either stop tuberculin altogether or keep the patient for a long period upon the concluding dose at certain extended intervals, the latter ranging from two to four weeks. My maximum dose has seldom been over one 0.1 mg. or 0.2 mg. of solid substance.

The intervals of doses must also be a matter of the physician's judgment. I have, in the beginning doses given tuberculin as often as every alternate day, some every third day, others twice weekly and still others but once weekly. There can be no fixed rule for administering tuberculin, and any physician who treats every case according to the same rule will surely meet with disaster.

The dietetic and hygienic conduct of a case is, of course, of the highest importance. I need not dwell upon that phase of treatment in this paper. Two points, however, should be impressed upon you; first, that all acute cases should, in the beginning of this treatment, have a rest of at least two months

in bed; second, that during the ambulant stage of treatment, and even after his discharge, the patient's exercise should be carefully regulated and the dangers of auto-inoculation from overstrain should be thoroughly ingrained into him. Many a cure has failed through neglecting to observe these two, which in my humble opinion are most important steps in the care of tuberculous patients.

To summarize: Of my series of twenty-eight cases, the ages ranged from nineteen to fifty-one years, the period of treatment from two and one-half months to about one year. The result: apparently cured nine cases, arrested six cases, improved eight cases, and not improved three cases, died two cases. There were then of the twenty-eight cases, fifteen apparently cured or arrested.

These results justify me in continuing along the same line of treatment and I hope to add very materially to this series. My small experience to the present time convinces me that bovine tuberculin in the treatment of pulmonary tuberculosis is most efficacious, as a remedial adjunct, and I hope in time to help prove that as a curative aid, it is superior to the human tuberculin. In the meantime, let us not forget that no tuberculin is a specific—that we sincerely hope, in time, some form will prove so—and that all we can attempt to do with our present knowledge is to stimulate the body's natural defenses and to aid the forces which nature has given us to combat tuberculosis.

SCIENTIFIC REVIEW

SYMPTOMOLOGY OF INFANTILE PARALYSIS.

Probably no disease presents a more interesting clinical picture than the so-called acute anterior poliomyelitis of children, and although the ordinary textbook case is familiar to most of us, many articles have been published in the past few years about other symptoms of the disease arising from damage to the higher centers and called polioencephalitis.

Wickman, "Beitrage zur Keuntnis der Heine-Medinschen Krankheit," Berlin, 1907, divided these cases into the following types:

- (1) Spinal poliomyelitis, common text-book type.
- (2) Ascending myelitis. The inflammation rapidly ascending until it affected the basal nuclei, and resembling Laudray's paralysis.
- (3) Bulbar or pontine cases, generally fatal.
- (4) Encephalitis, affecting the cerebral cortex.
- (5) Ataxia (cerebellar), resembling Friedreich's ataxia.
- (6) Polyneuritis, causing great local tenderness, and severest in the middle of the course of the nerve.
- (7) Meningitis, cases which develop before the onset of paralysis, with symptoms of meningeal irritation, usually passing off fairly soon.
- (8) The abortive form, where no paralysis de-

veloped, although constitutional symptoms were present resembling those of other patients who were subsequently paralyzed. To those forms must be added the spino-rubral form with acute tremor, described later by Muller.

In an epidemic recorded by Pasteur in 1896, a whole family of seven children were attacked with fever within two days. In three of these cases the fever was followed by paralysis, and by other nervous symptoms in four others. The eldest, age eleven, five days after the onset of the fever, was paralyzed in the left arm, and had a transient paralysis of the right side of the face and palate; the second child suffered from a rigid hemiplegia of the right side; another after a few days of fever was seized with tremors lasting a few days, and the youngest also had a strabismus.

In the experimental work done by Flexner and Lewis (Journal of Experimental Medicine, March 14, 1910), eighty-one monkeys were successfully injected with the virus, all resulting in paralysis, and some interesting conclusions as to the cause of the infection were made. They state that the cause of the lesion is no doubt a minute organism, which does not exist apart from its host, which becomes implanted upon the leptomeninges, especially in the region of the spinal cord and medulla, setting up changes most marked in perivascular lymph spaces of the arteries entering the anterior part of the cord. They also state that the brain is less commonly affected, but at times there is a paralysis of the cranial and facial nerves, and also in parts of the brain which do not respond by paralysis. Levaditi and Landsteiner (Compt. rend. Soc. de Biol., 1910, XVIII, 417) conclude, after their experiments on monkeys, that the virus enters through the nasal mucous membrane, and obtains entrance to the brain by following the branches of the olfactory nerve. Both Flexner and Levaditi conclude that the poison of the disease is excreted by the nasal mucosa, and suggest nasal discharges as possible sources of infection.

Many of the symptoms caused by the invasion of the mid-brain and its connections are never seen by the physician, because of the fact that the children are not often brought to him in the acute stages of the disease, and these symptoms often pass off quickly. A most interesting symptom is the so-called acute tremor, described by Gordon Holmes "On Certain Tremors in Organic Cerebral Lesions" (Brain, 1904, p. 327), and later by Miller, "On Certain Cases of Acute Tremor Occuring in Children" (Brain, 1909, pt. CXXV, vol. XXXII, p. 54). Miller showed that in several cases where the higher centers were affected a tremor came on